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Intracranial intricacies: Comprehensive analysis of rare skull base meningiomas—A single-center case series

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Key Clinical Message

This study paper's main goal is to report rare cases of skull base meningiomas that exemplify the complexities of diagnosis, therapy, and postoperative care. By describing these rare cases, we hope to advance knowledge of the clinical signs, difficulties, and prognoses of skull base meningiomas in a challenging anatomical setting. In the posterior cranial fossa, our investigation reveals a unique example of skull base meningioma that involved numerous cranial nerves and complex vasculature. A variety of visual abnormalities were present in the patient's clinical presentations, highlighting the wide range of symptoms that these tumors might cause depending on their precise positions. These cases highlight the critical importance of preoperative imaging, including high-resolution MRI and angiography, as well as the diagnostic difficulties these tumors pertain. By reporting these instances, our research adds to the body of knowledge about skull base meningiomas and offers insightful information about the nuances of their therapies. Our findings highlight the importance of individualized treatment plans, interdisciplinary cooperation, and the demand for continued study to better comprehend these convoluted tumors. Such studies are essential for advancing our knowledge of these enigmatic tumors, guiding clinical judgment, and eventually improving patient outcomes. These findings are important because they can fill information gaps, improve treatment plans, and encourage additional research in neuro-oncology.

Abstract

This study presents a series of three rare cases of skull base meningiomas, emphasizing the complexities in diagnosis, treatment, and postoperative care. The patients' clinical presentations and imaging highlighted the diverse symptoms and challenges associated with these tumors, found in intricate anatomical locations. The cases underscore the crucial role of preoperative high-resolution imaging and angiography in diagnostic accuracy. Surgical intervention, guided by a multidisciplinary approach, is pivotal in managing these demanding cases. Histopathological examinations confirmed atypical meningiomas. The

postoperative phases involved meticulous care to ensure optimal recovery and functional outcomes. Our findings contribute to the understanding of skull base meningiomas, emphasizing the need for personalized treatment plans and ongoing research to improve patient outcomes in neuro-oncology.

KEYWORDS

anatomical locations, angiography, clinical presentation, diagnosis, high-resolution imaging, imaging, multidisciplinary approach, postoperative care, skull base meningiomas, surgical intervention, treatment

1 | INTRODUCTION

Meninges are the protective tissue layers that coat the brain and spinal cord, and a skull-based meningioma is a specific kind of brain tumor that develops from them. Meningiomas are typically benign (noncancerous)[1] slow-growing entities, but because of their location and the pressure they put on nearby brain regions, they can nonetheless have serious health consequences. Meningiomas make up 13%–26% of all primary intracranial tumors, making them the most prevalent. Meningiomas of the skull base make up about 25% of all meningiomas, the reported distribution was in the calvarial-to-skull base ratio of 2.3:1. In the United States, the annual incidence of these tumors is estimated to be 6.0 per 100,000 people per year.[2] Moreover, the incidence and prevalence of skull base meningiomas in the general population can vary based on several factors, including age, gender, and ethnicity. Skull base meningiomas can afflict people of all ages, however, they typically show up in adults. The age range of 40–70 is where the highest incidence rates are seen.[3] The obvious gender imbalance in skull base meningiomas is one of their most prominent characteristics. Studies consistently show that women are diagnosed with these tumors more often than males. The gender bias in certain kinds of meningiomas is very pronounced.[4] By their very nature, skull base meningiomas are found close to several important structures. These include the brain stem itself, the major blood arteries, and the cranial nerves incharge of vision, hearing, balance, and facial functions. As a result, there is a higher risk of neurological abnormalities and serious functional impairment. Accurate localization and characterization of these tumors need the use of sophisticated imaging techniques, such as computed tomography (CT) and magnetic resonance imaging (MRI) scans.[5] It might be challenging to discern between a tumor and normal structures, particularly in the delicate area of the skull base. The major method of treating skull base meningiomas is still surgery. The surgical removal of skull base meningiomas can result in a number of postoperative problems, including imbalance issues, hearing loss, facial numbness, and visual impairment. Concerns include the possibility of hematoma, infections, and cerebrospinal fluid leaks. To handle these difficulties, careful postoperative monitoring and rehabilitation techniques are essential.[6] Skull base meningiomas provide an intriguing glimpse into the field of histopathology by illuminating their origin, specific patterns, and the vital implications these traits have for clinical care. Their genesis from arachnoid cap cells, specialized arachnoid mater cells,[7] and the important layer of tissue that surrounds the brain and spinal cord are at the core of their histological identity. Arachnoid cap cells contribute to the complex balance of the central nervous system by controlling cerebrospinal fluid (CSF) dynamics. Arachnoid cell growth and the fibrous tissue they produce are what cause the whorling pattern to appear. Additionally, there are several histological subtypes of skull base meningiomas, including meningothelial, fibrous, psammomatous, transitional, atypical, and anaplastic variations. Each subtype carries unique features that have an impact on the prognosis and course of treatment. Meningiomas are divided into three main types according to the WHO classification of CNS tumors, which is reflected in the WHO grades I (benign), II (intermediate), and III (malignant).[8] Meningiomas are subdivided in 15 histological and cytomorphological variants of which nine variants correspond to WHO grade I, three variants correspond to WHO grade II, while another three variants correspond to the malignant type of WHO grade III meningiomas.[8] Atypical and anaplastic meningiomas, which have enhanced cellularity, nuclear atypia, and significant mitotic activity, are at the other end of the range.[7] While atypical and anaplastic subtypes of meningiomas provide higher problems because of their aggressiveness,[9] needing more rigorous treatment options including surgery, radiation, and targeted medicines, meningothelial meningiomas are the most frequent and frequently show a favorable prognosis. The tendency of skull base meningiomas to cause neurological impairments is one of their defining characteristics. As a result of the tumor's compression or relocation of important brain regions, these can cause motor deficits, characterized by

weakness or paralysis in the limbs, sensory impairments, such as numbness or tingling. Skull base meningiomas are particularly prone to compressing the cranial nerves that regulate face movement, swallowing, vision, hearing, and other bodily processes. Depending on the afflicted nerves, cranial nerve impairments can show up in several different ways. Patients with skull base meningiomas frequently have headaches as a symptom. These headaches are reported to be chronic, dull, and concentrated at the tumor's location. Increased intracranial pressure brought on by the tumor's existence is most likely the mechanism causing these headaches. Positional changes or activities that alter blood flow and cerebrospinal fluid dynamics can make the headaches worse. Patients with skull base meningiomas experience symptomatology variations that are mostly determined by the location and size of the tumor. The front, middle, and posterior portions of the skull base each include unique structures that are in charge of carrying out diverse duties. As a result, the symptoms vary depending upon on which structures are impacted.[10] Skull base meningiomas have a complicated clinical landscape that calls for a thorough and individualized approach to therapy. In the management of skull base meningiomas, surgical intervention continues to be a mainstay, especially when the tumor is producing neurological impairments, substantial symptoms, or is amenable to removal without putting important structures at undue risk. The extent of resection is determined by the size, location, and general health of the patient, among other things. The best possibility of long-term control and a potential reduction in the requirement for adjuvant medicines are provided by complete resection, which is the desired outcome. Skull base meningiomas that cannot be surgically removed because of their position or the patient's health can be treated with radiation therapy, which includes methods like stereotactic radiosurgery. Radiation therapy targets the tumor with strong doses of radiation while preserving the surrounding healthy tissue. For persistent or recurrent tumors in particular, it can successfully suppress tumor growth. Particularly for small, asymptomatic skull base meningiomas, observation, or "watchful waiting" may be useful in some circumstances. Regular imaging is done to track the growth of the tumor over time, and treatment is postponed until symptoms or progression are evident. Close monitoring is necessary to guarantee prompt intervention when necessary.[5] Skull base meningiomas are complex tumors and controlling them effectively calls for a multidisciplinary approach because of the wide range of expertise needed. Working together, neurosurgeons, radiation oncologists, neurologists, otolaryngologists, ophthalmologists, and other specialists choose the best course of action for each patient's particular needs. The location, size, potential impact on vital functions, and general health of the patient are all thoroughly assessed thanks to this cooperative effort. Treatment decisions, whether they require surgery, radiation therapy, or observation, are aided by multidisciplinary consultations. A well-informed, patient-centered treatment plan that maximizes the likelihood of positive outcomes while minimizing potential consequences is made possible thanks to the knowledge of each specialist.

2 | METHODS

This retrospective single-center case series was conducted at the Shaheed Mohtarma Benazir Bhutto Trauma Center in Karachi, focusing on a rare cohort of patients diagnosed with skull base meningiomas. Patient selection was based on the rarity of the condition, emphasizing the complexities in diagnosis, treatment, and postoperative care. Detailed clinical data, encompassing demographic information, clinical presentations, imaging findings, histopathological results, surgical details, and postoperative care, were meticulously collected from the medical records and files of the patients. Imaging analyses were performed by experienced radiologists to evaluate preoperative imaging, including computed tomography (CT) scans and magnetic resonance imaging (MRI). These analyses played a pivotal role in determining the location, size, and characteristics of the meningiomas, guiding surgical planning and approach for each case. Histopathological examinations were conducted on biopsy or excision specimens obtained during surgery, with skilled pathologists determining the histological subtype and grade of the meningiomas based on the World Health Organization (WHO) classification. Surgical interventions were performed by a multidisciplinary team of neurosurgeons, tailoring the surgical approaches and techniques to each case based on the tumor's specific characteristics and location.

2.1 | Case 1

The patient was a 30-year-old male without any significant previous medical comorbidity, who presented to the clinic with complaints of aphasia and weakness on the whole right side of his body. The symptoms were gradual in onset and worsening. On examination, the Glasgow Coma Scale score was 15/15 when he visited the clinic, although possibly less movement on the right side. A plain CT scan of the brain was carried out; images were obtained in multiple planes. He was diagnosed with left sphenoid wing meningioma shifting to the midline. In a computed tomography (CT) scan of the left frontal lobe, a well-defined extra-axial mass lesion measuring 6.2 × 5.6 × 5.5 cm (AP × TR × CC)

was seen. It was accompanied by severe perilesional edema that compressed the brain parenchyma underneath and effaced the frontal horn of the ipsilateral lateral ventricle, creating a midline shift of 1.0 cm on the right side with no change in interval. The structures of the posterior fossa appeared to be unremarkable (Figure 1). The mastoid air cells and paranasal sinuses could be seen as normal. MRI of the patient is shown in Figure 2.

After completing the necessary examinations, the patient was hospitalized and underwent a biopsy after 12 days. The patient was prepped before the procedure began and general anesthesia (100 cc) was administered. A pterional craniotomy was performed to gain direct access to the tumor within the sphenoid wing, a thick-walled abscess was present, the abscess was excised, hemostasis was established, duraplasty with pericranium was performed, the wound was cleaned with H_2O_2 and gentamicin, bone was placed and fixed, the wound was then securely bound up and closed. Though the tumor was too invasive to remove completely, a near-total resection was accomplished. The histopathological classification of the tumor was a sphenoid wing meningioma with features consistent with Atypical Meningioma WHO Grade II. The biopsy consisted of multiple tan-white tissue pieces, measuring 3.6 × 2.4 cm. The cut surface was tan-white. Microscopy revealed a neoplastic lesion composed of Meningothelial cells arranged in whorls along with some places showing increased cellularity with a focal diffuse pattern of growth. Individual cells had moderate amounts of eosinophilic cytoplasm and vesicular chromatin. The lesion was invading the adjacent glial tissue. Necrosis was also identified. Scattered mitotic activity was seen (4 mitoses 10 HPF). Marked psammomatous calcifications were also present.

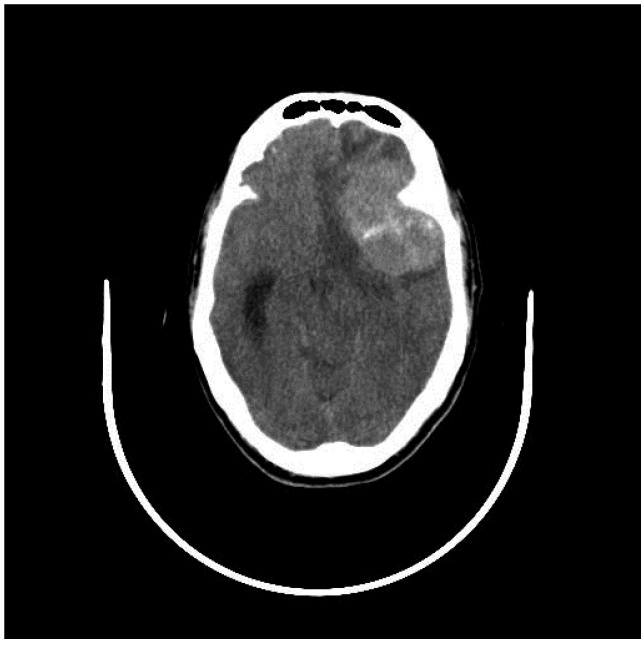

FIGURE 1 CT Scan showing well-defined extra-axial mass lesion with perilesional edema and midline shift in left frontal lobe.

After 2 weeks, the patient was discharged with prescriptions for amoxicillin, linezolid, naproxen, and ondansetron and was instructed to undergo an MRI after 1 month and make a follow-up appointment at the clinic after 1 week.

2.2 | Case 2

A 60-year-old female patient, with no known comorbidities, presented to the emergency ward with complaints of vision loss in her left eye since last year, characterized by darkening of vision and loss of color vision without diplopia or selective field loss. Additionally, she had been experiencing severe, localized left eye headache persisting for 3 months. The headache was sudden in onset, aggravated by noise, and relieved by intravenous analgesics. Furthermore, for the past month, she reported fits leading to unconsciousness with uprolling of eyes and tongue biting, along with episodes of paresthesia. These episodes were characterized by abnormal sensations, such as tingling or numbness, experienced in various parts of her body.

On neurological examination, she showed intact cranial nerves. Her visual acuity was measured 6/6 for both the right and left eyes, but perception of light (NPL) was absent in the left eye. On the examination, her Glasgow Coma Scale score was E4M6V1 (aphasic) with right-sided hemiparesis, she exhibited a power of 0/5 in her right upper and lower limb, indicating complete paralysis, while her left upper and lower limbs demonstrated full power (5/5). The reported left-sided vision loss accompanied by headache and blurred vision, necessitated a fundoscopy whose result showed a pale white disc in the left eye with compressive optic neuropathy. A cardiovascular examination revealed a blood pressure of 148/92 mmHg and a heart rate of 89 beats per minute, with regular ECG rhythms. Her respiratory parameters were stable with 100% oxygen saturation. The gastrointestinal system revealed a soft and tender abdomen with an NG tube and foley catheter in place. Following these clinical presentations, various investigations were ordered, including complete blood count (CBC), angiography, X-ray, CT scan, and MRI. The CT scan of the brain revealed a well-defined extra-axial homogenously hyperdense lesion in the left frontotemporal area, measuring 5.5 × 5.0 × 5.5 cm (AP × TR × CC), as shown in Figure 3. This lesion was associated with surrounding edema and was causing mass effect. Additionally, it resulted in the effacement of the ipsilateral lateral ventricle and a midline shift of about 0.8 cm to the right side. There was no evidence of hydrocephalus, and the basal cisterns and extra-axial spaces were preserved. The posterior fossa structures appeared unremarkable, while

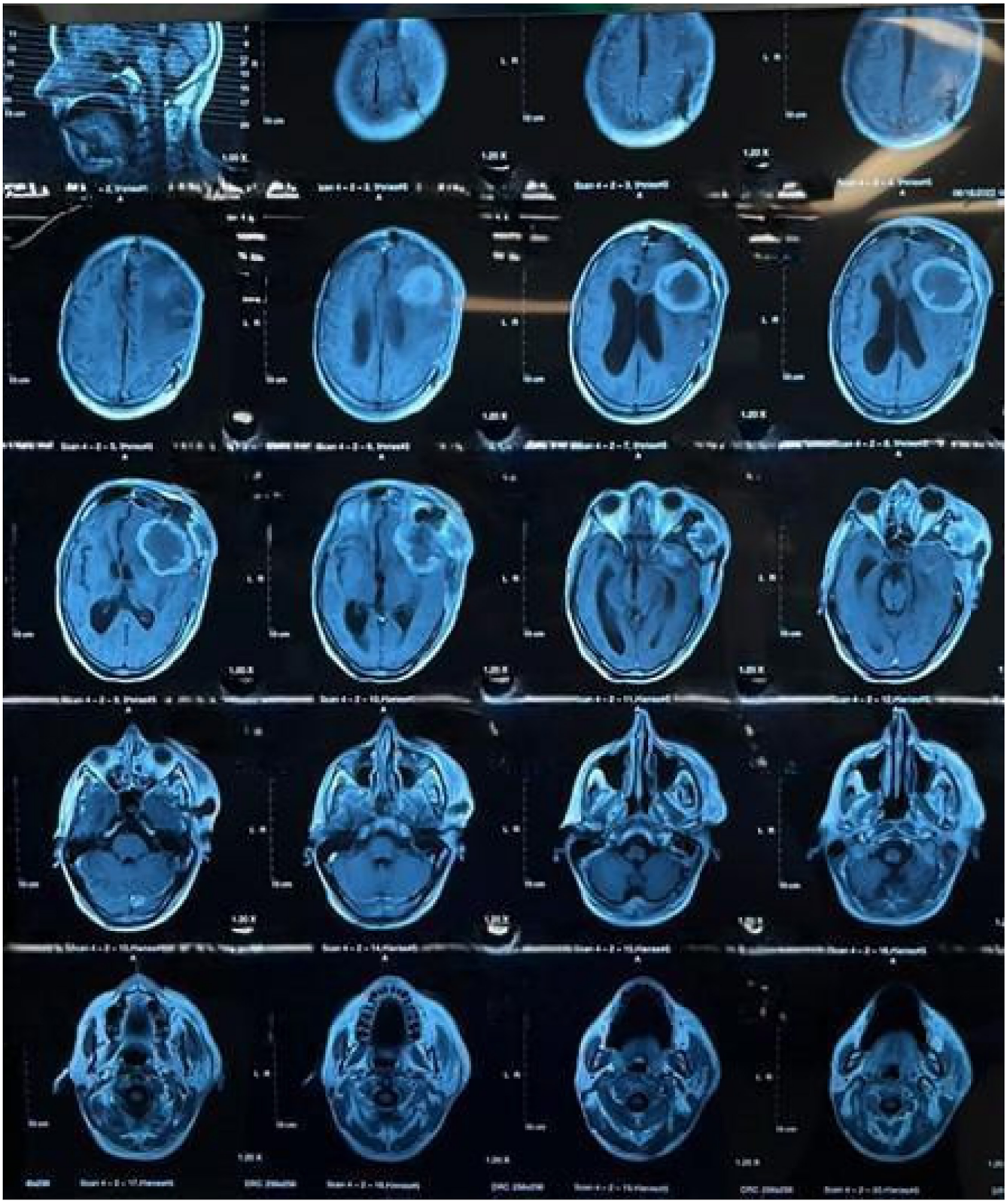

FIGURE 2 MRI of the patient.

the paranasal sinuses and mastoid air cells were found to be normal. MRI of the patient is shown in Figures 4 and 5.

Before proceeding to surgery, digital subtraction angiography (DSA) was performed using the Seldinger technique to visualize the blood supply to the tumor. The angiography revealed that the tumor's blood supply originated from the middle meningeal branches of the left maxillary artery. To obstruct the blood flow to the tumor

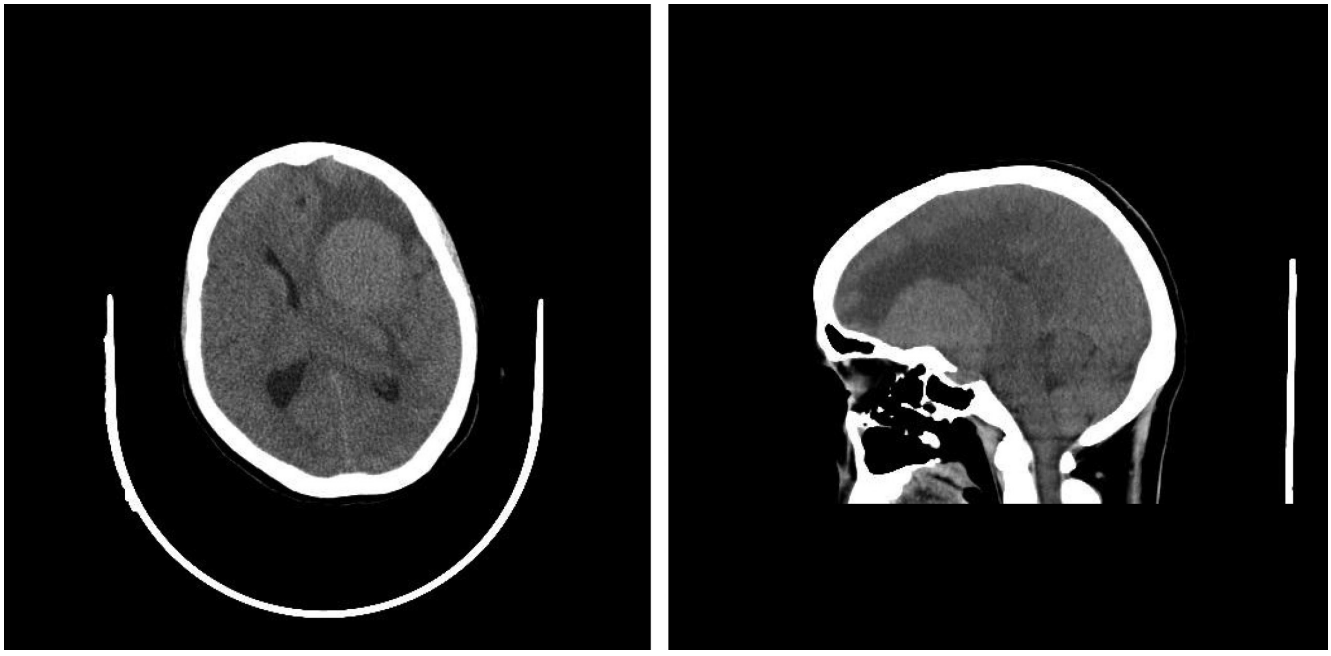

FIGURE 3 CT scan displaying a well-defined extra-axial homogenously hyperdense lesion at the left frontotemporal area, accompanied by surrounding edema. The lesion is causing mass effect, evident by the effacement of sulci along with compression of the ipsilateral lateral ventricle and a midline shift of approximately 0.8 cm to the right side.

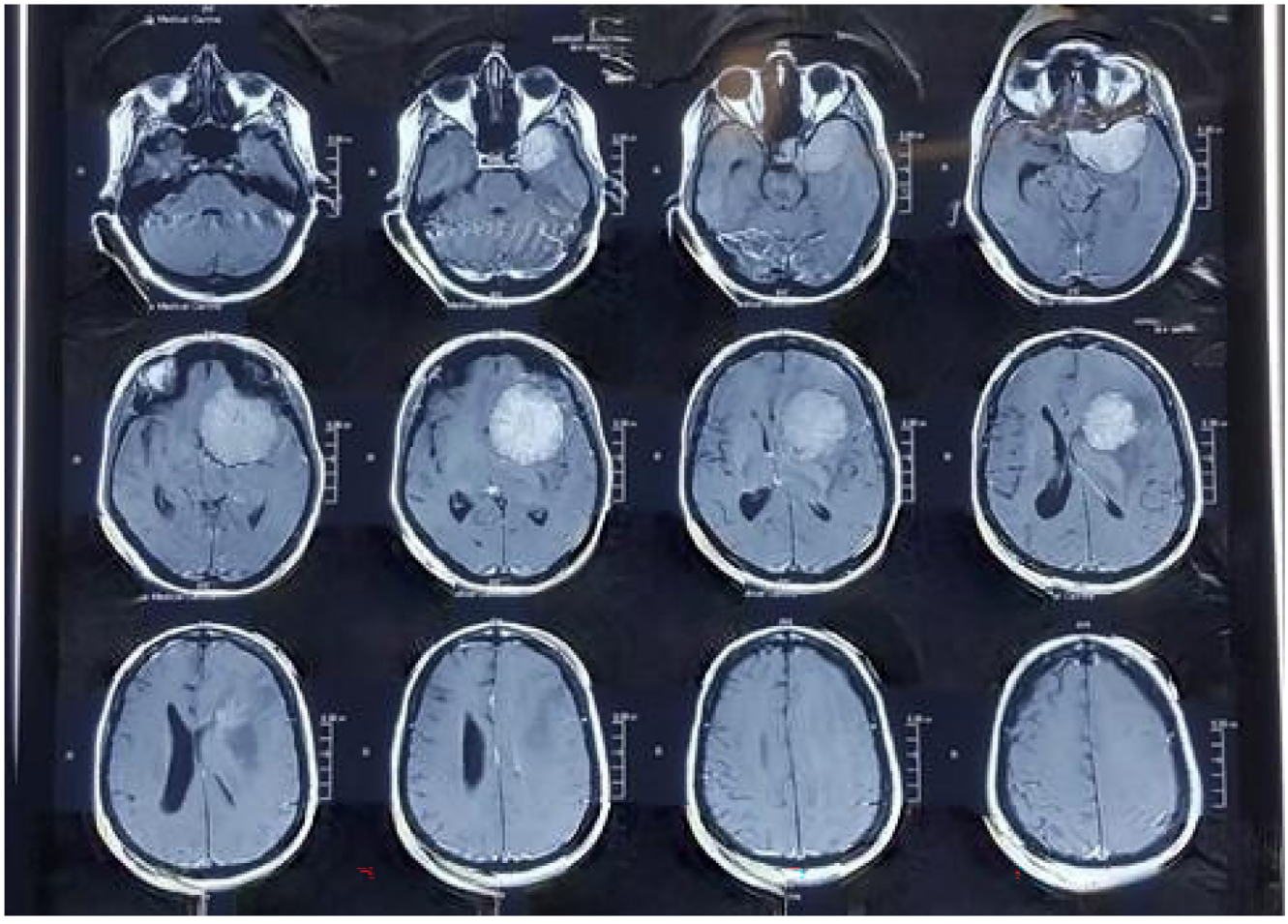

FIGURE 4 Preoperative axial magnetic resonance imaging brain of a patient with a large left sphenoid wing meningioma.

and facilitate the surgical procedure, the blood supply was selectively cannulated with a microcatheter, with successful embolization of the left middle meningeal artery using gel form. The postembolization angiogram demonstrated significant occlusion of the tumor vasculature. The embolization procedure was successful in blocking the blood flow from the left middle meningeal artery using gel foam. She was advised to immobilize her right leg for 6 h following the DSA procedure. Hemostasis was secured during the procedure, and no intraoperative complications were observed.

Medical investigations revealed that she had a brain tumor located in the area of the sphenoid wing that required immediate surgical intervention. In January 2023, she was admitted to the neurosurgery ward at Shaheed Mohtarma Benazir Bhutto Hospital where she underwent an excision biopsy. During the procedure, the patient was prepared and draped in a spine position with the head tilted to the right side. A pterional flap was raised, followed by a craniotomy. The dura was opened, and the tumor was identified. The surgical team performed a near-total resection of the tumor, ensuring hemostasis was secured. Duroplasty was done, and the closure was performed in layers. Additionally, an ASD (artificial subdural space) was applied.

The operative findings revealed a sphenoid wing meningioma that was firm, solid, and nonsuckable, measuring around 6×6 cm, with a vascular extra-axial lesion. It was located at the middle cranial fossa, adherent to the medial wing of the sphenoid bone, and was abutting the left internal carotid artery. Despite the complexity of the case, a successful total excision of the sphenoid wing meningioma was achieved. The patient's condition was closely monitored in the ICU postsurgery. On examination, her vital signs were relatively stable, with a blood pressure of 129/68 mmHg and a pulse rate of 97 beats per minute.

Brain CT scans revealed signs of decomposition while dressing changes were clean and dry. The management plan included weaning her off the ventilator and encouraging mobilization as tolerated. The patient was permitted sips of fluids orally and received NG feeds. She received intravenous medications, including Ceftriaxone, Enalapril, Mannitol, Dimenhydrinate, Dexamethasone, Omeprazole, and Nimodipine. Follow-up instructions included continuing intravenous medications until she was able to take oral intake.

She was discharged from the hospital on January 25, 2023. The multidisciplinary team of neurosurgery and ophthalmology ensured appropriate postoperative care and follow-up. Further investigations and monitoring were planned to assess her recovery progress. The patient received the necessary medical attention to address her vision loss and headache complaints, and her progress was closely monitored during follow-up visits.

2.3 | Case 3

In July 2022, a 25-year-old male patient presented to the outpatient department (OPD) with a complaint of numbness in his arms and legs persisting for the past 1.5 months. Based on the nature of his symptoms and suspected neurological implications, he was advised to be admitted to a neurosurgery ward for further examination and management.

On examination, all cranial nerves were found to be intact. Sensory examination indicated intact sensation to pinprick in the upper limbs and intact sensation in the lower limbs. Grip strength in the right upper limb was noted as 4/5. He had normal flexion and extension of both limbs with a positive Hoffmann response.

A CT scan was conducted using various planes and appropriate window settings, revealing a distinct solid lesion situated within the infratentorial posterior fossa near the foramen magnum. This extramedullary lesion measured approximately 3.0×4.2×3.9 cm, exhibiting close proximity to and compression of the brain stem, pons, and medulla anteriorly, as well as the bilateral cerebellar hemispheres (Figure 6). It also abutted the cerebellar vermis superiorly and the occipital bone and foramen magnum posteriorly, though no evidence of bony erosion was evident. The lesion extended about 1.3 cm below the foramen magnum. Ventricles remained intact, and no signs of hydrocephalus were detected. The paranasal sinuses and mastoid air cells showed normal appearances, while bilateral middle and inferior turbinate mucosal thickening was noted. The recommended course of action involved further investigation through biopsy. MRI of the patient is shown in Figure 7.

In September 2022, the patient underwent an excisional biopsy. During the procedure, the patient was prepared and draped in a prone position. A midline vertical incision was made from the occipital protuberance down to C2, and a flap was raised, followed by a craniectomy of approximately 3×3 cm. C1 laminectomy and partial C2 laminectomy were performed. Upon opening the dura, the tumor was identified, exerting anterior compressive pressure on the spinal cord. Gross total excision of the tumor was achieved, hemostasis was secured, duroplasty was performed, and closure was done in layers. An artificial subdural space (ASD) was created.

The findings indicated the presence of a firm, nonsuckable vascular extramedullary lesion, measuring around 5×4×3 cm. This lesion extended from the foramen magnum to C2, causing anterior compression of the spinal cord. Histopathological examination of the biopsy specimen revealed a cellular neoplastic lesion with syncytial and meningothelial whorls. The cells exhibited ovoid to elongated nuclei with eosinophilic to clear cytoplasm. Up

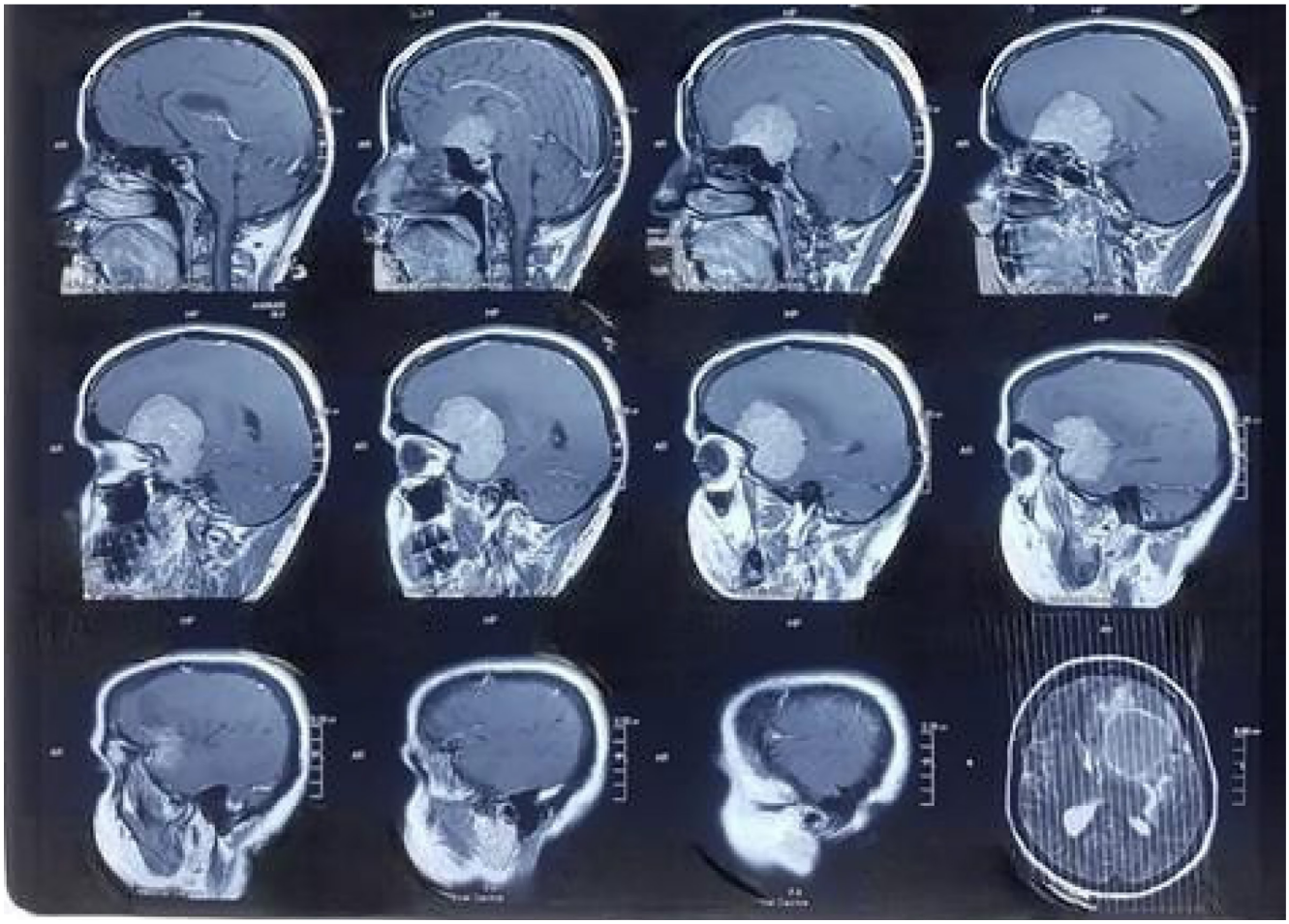

FIGURE 5 Preoperative sagittal magnetic resonance imaging brain of the patient.

to 5–6 mitotic figures per 10 high-power fields (HPF) were observed, with no prominent psammomatous calcifications. The diagnosis based on the biopsy specimen was foramen magnum meningioma, favoring atypical meningioma of WHO grade II.

In the postoperative phase, the patient was kept NPO for 6 h. Blood sugar levels were monitored and managed with regular RBS (random blood sugar) checks and medications. A repeat CT scan of the brain was performed. Intravenous fluids (normal saline), ceftriaxone, paracetemaol, dimenhydrinate, dexamethasone, ketorolac tromethamine, Omeprazole, and Calcium + Vitamins D3 + C + B6 were administered as part of postoperative care.

The patient's postoperative recovery was satisfactory, and he was discharged in October 2022. Close follow-up was planned to monitor his progress and ensure optimal postoperative care.

3 | DISCUSSION

Meningiomas are the most prevalent type of primary central nervous system tumor in adults, accounting for 55% of all nonmalignant tumors in the United States.[11] They are more prevalent in the supratentorial compartment, which is home to the most common cranial vault tumor. Supratentorial meningiomas are categorized as non-skull base or skull base, with distinctions in clinical presentation, tumor grade, prognosis, and treatment approaches.[12]

When discussing prognosis and care with patients, practitioners must understand the particular features of supratentorial NSBM. The current clinical decision-making and contemporary surgical and adjuvant therapy of supratentorial NSBM are discussed in this study.[13]

Meningiomas are often slow-growing benign tumors that develop from the linings of the brain (the meninges) near the base of the skull. These tumors can emerge from a variety of locations inside the skull base.[14] Skull base meningiomas are among the most difficult pathologies seen by neurosurgeons due to their depth, invasion, vascularity, texture/consistency, and connection to bone structure, cranial nerves, and blood arteries. In order to minimize brain retraction and ideally identify, protect, manage, and manipulate delicate neurovascular systems, resection of complicated skull base meningiomas frequently necessitates significant bone removal to enable

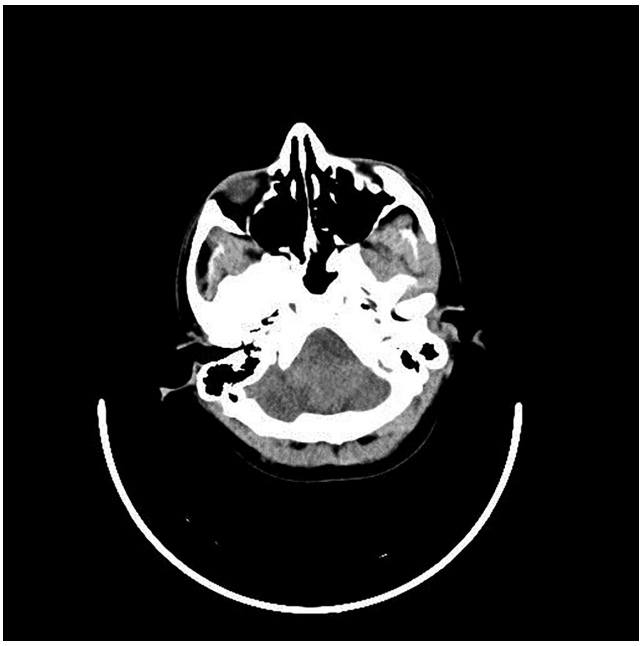

FIGURE 6 CT scan demonstrating a well-defined solid lesion at the infratentorial posterior fossa, located at the extramedullary region near the foramen magnum. The image reveals features suggestive of an infratentorial posterior fossa neoplastic lesion with a defined extent and associated adjacent mass effect.

appropriate exposure of the tumor and surrounding area. To handle difficult skull-base tumors, a range of conventional skull-based therapies have emerged, with meningiomas serving as the paradigm in terms of both complexity and frequency.[15] Meningiomas originate from arachnoid cells, especially those found in the arachnoid granulations that coat the brain and spinal cord. Following the closure of the neural tube in the embryo, these cells grow along a ventral-dorsal axis. The meninges play an important role in brain and skull development by secreting substances that promote neuronal division, migration, and maturation. The meninges' cellular origin varies depending on location, with dorsal and posterior portions derived from mesoderm and ventral regions derived from migrating neural crest populations. These distinctions might explain clinical connections linked to the meningioma site, such as the enrichment of certain genetic driving events and long-term behavior. The role of CNS lymphatics inside the meninges is expanding, with anatomic variation driven by phylogeny and physical variables such as high CSF pressure. This lymphatic system governs CNS immune surveillance, which may have an influence on therapeutic treatment. Extracellular matrix remodeling genes and meningeal lymphatic genes are expressed in immune-enriched subtypes of meningioma. To treat molecularly characterized meningioma subtypes, a personalized strategy that includes meningeal lymphatics and their function in immune control may be required.[16] Meningiomas include molecular subgroups with unique driver mutations and clinical characteristics, according to genomic research. The most prevalent change is the biallelic deletion of the tumor suppressor NF2, which is more common in non-skull base regions. Recurrent mutations in SMARCB1, which is situated on chromosome 22q, are seen in certain NF2 mutant meningiomas. Other subgroups are focused on neural crest-cell-derived skull-base lesions, which involve PI3K and hedgehog signaling pathway activating variations.[17,18] The clinical appearance of individuals with symptomatic skull base meningiomas differs depending on the tumor location, mass effect, and cerebral edema. Patients with elevated intracranial pressure may present with either localizable symptoms (focal neurological impairment or seizures) or nonlocalizable symptoms (positional headaches, nausea/vomiting, diplopia, and somnolence). Seizures are one of the most common symptoms in individuals with symptomatic meningiomas, involving around 10%–70% of supratentorial NSBM patients. Seizures are strongly predicted by the presence of peritumoral edema. Other symptoms include vision problems, hearing loss, balance issues, facial numbness or paralysis, difficulty swallowing, and more.[19–21] Systemic therapies are presently reserved for meningiomas that are complicated, progressive, and recurring and cannot be addressed with alternative treatments such as surgery or radiation. Despite the lack of strong proof of efficacy, research into systemic treatments for this patient population continues due to significant unmet demand. Multiplatform analysis of 115 meningiomas[22] revealed that therapies targeting NF2 and topoisomerase IIA would help the vast majority of meningioma patients. The epidermal growth factor receptor was the most often targeted protein, and antibody-drug conjugates might be investigated. To address particular tumor aberrancies, current investigations are driven by a mechanistic approach and preclinical modeling.[23,24]

Meningiomas are classified into Grades I through III by the World Health Organization (WHO) based on their histological features. Grade I is the least aggressive, with well-defined limits and modest development. Certain histological subtypes, on the contrary, are associated with poorer clinical outcomes and are classified as WHO grade II or III, suggesting malignancy. The most prevalent subtype is meningothelial meningioma, which is followed by fibrous and transitional forms. These three subtypes account for over 80% of all meningiomas diagnosed by radiologists as typical meningiomas. In comparison, each of the other histological subtypes accounts for just 1%–3% of all meningiomas.[25] Grade II is more aggressive than grade I with characteristics such as increased cellularity and mitotic activity. With substantial

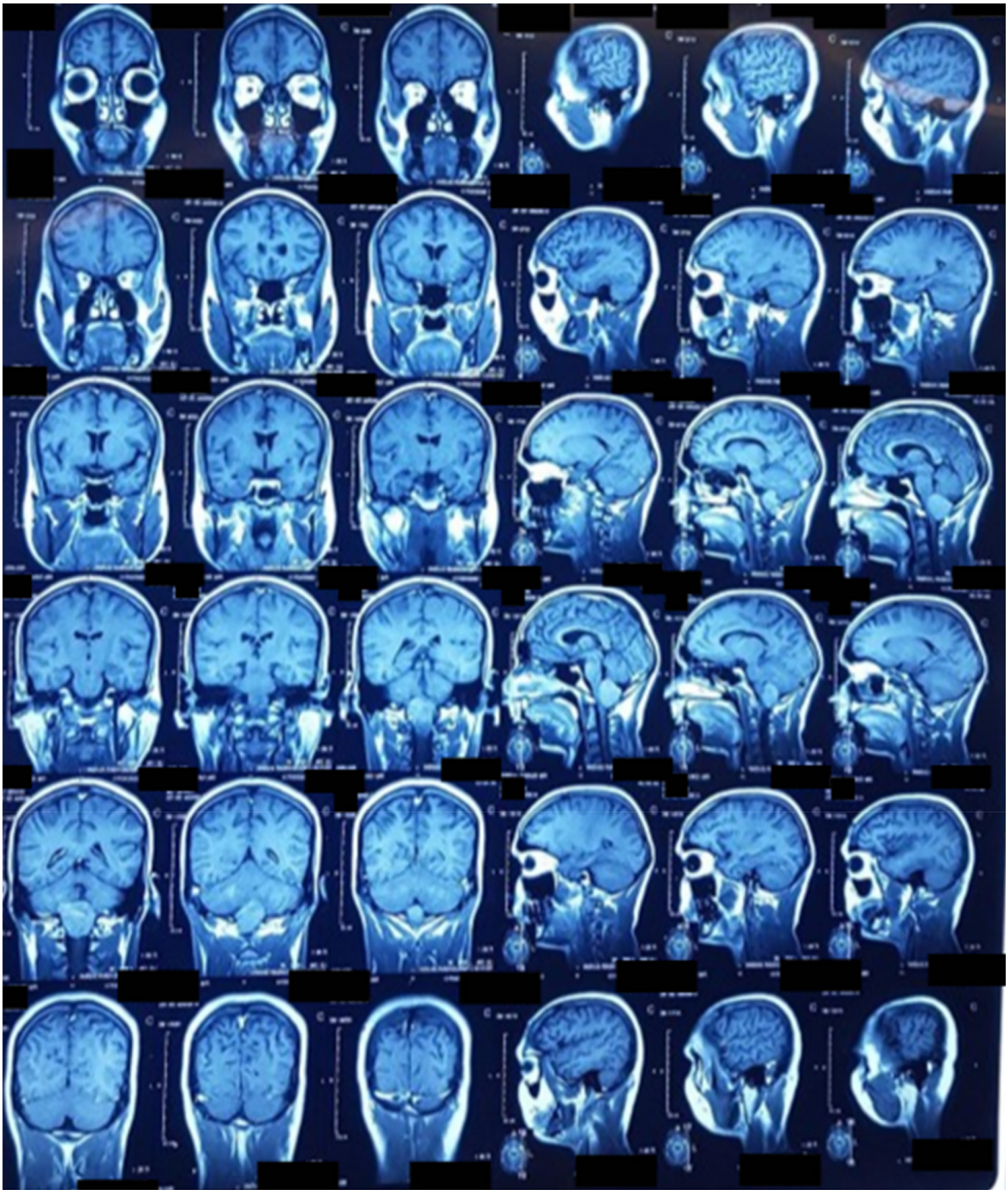

FIGURE 7 MRI of the patient.

cellular atypia and strong mitotic activity, Grade III is the most aggressive and has the poorest prognosis. Surgery, radiation therapy, and chemotherapy are common treatments for Grade III meningiomas.[26] The grading of a meningioma is critical for selecting the best treatment approach and predicting the patient's prognosis. A pathology report or biopsy typically offers information on the grade of the meningioma, which helps guide treatment decisions. Overall, the WHO grading system aids in the identification of effective treatment for meningioma.

Meningiomas often appear as sessile or lentiform, well-circumscribed extra-axial mass lesions with broad-based dural attachment. They often show hyperdensity on noncontrast CT, iso- to hypo intensity on T1-weighted scans, and iso- to hyperintensity on T2-weighted imaging. Meningothelial, fibrous, and transitional meningiomas can have varying intensities on diffusion-weighted images, causing apparent diffusion coefficient (ADC) values to fluctuate substantially. When compared to the brain parenchyma, most of them will show no diffusion restriction or facilitation on diffusion-weighted imaging.[27] When the tumor grows, it might cause peritumoral brain edema. Meningiomas might emerge having sunburst or spoke-wheel patterns on transcatheter angiograms. These frequent imaging abnormalities are most likely due to meningioma histological subtypes. As a result, some of these imaging findings may or may not be applicable to unusual histological variations of meningiomas. The vast range of imaging results may lead to misdiagnosis of meningiomas.[28] Meningiomas with WHO grade II or III are associated with high recurrence rates and mortality. Successful gross total resection reduces the rate of recurrence significantly more than subtotal resection. In WHO grade I meningiomas, tumor consistency, associated with tumor histology, is an important factor in determining the extent of tumor resection and surgical outcome.[29]

Psammomatous meningioma is a histological variation with psammoma bodies that is classified as WHO grade I. It is located in the skull and intraspinal spaces, most notably the thoracic spine. CT scans indicate widespread calcification or calcification at the tumor's perimeter, with T1-weighted images displaying iso-intensity and low signals. The tumor appears low intensity on T2-weighted imaging, however, it might alternatively seem iso- to high intensity. On CT scans, MR pictures may reveal hypo intensities; however, calcifications are predicted to cause signal attenuation. ADC readings of psammomatous meningiomas are most likely within the normal range.[30] Angiomatous meningioma is a rarely seen variant of meningioma with a predominance of blood vessels, comparable to WHO grade I. Unlike most meningiomas, it may have a small masculine preponderance. The tumor appears hypointense on T1-weighted imaging, hyperintense on T2-weighted pictures, and somewhat hypointense on diffusion-weighted images. Postcontrast MR images reveal dramatic enhancement due to significant hypervascularity. Homogeneous enhancement is reported in 85% of instances, with the remaining 15% exhibiting heterogeneous enhancement or cyst development. Most of the time, internal flow voids are identified.[31] Other kinds of grade I meningiomas include Microcystic meningioma, Secretory meningioma, and lymphoplasmacyte-rich meningioma.[32] Clear cell meningioma corresponds to a WHO grade II tumor, distinguished by polygonal cells with transparent cytoplasm and abundant interstitial collagen. It is more frequent in children and is placed near the cerebellopontine angle. The tumor's density varies, with high density being common. Its ADC values may be comparable to those of ordinary meningiomas, and it frequently exhibits considerable contrast enhancement. Cysts are seen in 60% of patients, and 78% had peritumoral brain edema.[33] Anaplastic meningioma has a cytology that is malignant, resembling carcinoma or high-grade sarcoma, and hence corresponds to WHO grade III. Atypical meningioma has intermediate malignant characteristics between grade I and anaplastic meningioma and correlates to WHO grade II. In clinical terms, these subtypes are more prevalent in older individuals, with a male predominance, as opposed to normal benign meningiomas. Inconsistent tumor-brain interfaces, uneven tumor margins, inhomogeneous tumor appearances, and a lack of capsule-like enhancement at tumor margins are all common imaging characteristics.[34] Other histological variants of meningioma include metaplastic meningioma, choroid meningioma, and papillary and rhabdoid meningiomas. Metaplastic meningioma contains osseous, cartilaginous, myxoid, lipomatous, or xanthomatous tissue and can be localized or widespread. It is sometimes termed after prominent mesenchymal components, although whether it is genuinely a metaplastic form is still debated. The imaging properties differ depending on the mesenchymal components. Chordoid meningioma contains tissue comparable to chordomas and can manifest in children as Castleman's illness. Papillary meningioma has a perivascular pseudo-papillary appearance with uneven borders, heterogeneous enhancement, mild to significant irregular peritumoral edema, and cyst development. Rhabdoid meningioma is characterized by sheets of rhabdoid cells and has a dismal prognosis.[35–37] Meningiomas are tumors that develop from meningothelial or arachnoid cap cells, and they are more prevalent in places with a high cell density. Meningiomas are commonly formed in arachnoid granulations or villi, which include a significant number of arachnoid cap cells. Because these villi are clustered along the dural venous sinuses, they are commonly seen next to the sinuses. Convexity, parasagittal and falcine, sphenoid and middle cranial fossa, frontonasal, posterior fossa, and orbital meningiomas are all common intracranial or juxta cranial meningiomas. Intraspinal meningiomas account for around one-tenth of all intracranial meningiomas.[38] Ectopic meningiomas, which account for 1%–2% of all meningiomas, are uncommon and can be primary or metastatic.

They often begin in the skull and head and neck regions, with uncommon sites including the lung, mediastinum, retroperitoneum, pelvis, and extremities. Primary ectopic meningiomas are hypothesized to arise from arachnoid cells around cranial nerves entering the skull or to be spread during skull development. Other reasons for meningiomas at places other than the neuraxis include congenital, ectopic, meningothelial cell resting, and meningothelial differentiation from pluripotent mesenchymal cells. Imaging results vary, with intraradiploic meningiomas displaying both osteoblastic and osteolytic lesions, leading to differential diagnoses such as metastasis, multiple myeloma, osteosarcoma, fibrous dysplasia, and intraosseous hemangioma. Because of their rarity, ectopic meningiomas may not be the best working diagnosis before histological confirmation.[39]

3.1 | Diagnostic challenges

Skull base meningiomas have a unique subtype known as sphenoid wing meningiomas. They develop in the area of the sphenoid bone, which is found in the middle cranial fossa (a region at the base of the skull) and contains vital organs including the cavernous sinus and the temporal lobe of the brain. As a rare complication and overlapping with other conditions, sphenoid wing meningioma presented numerous challenges in diagnosis. Neuroimaging methods such as computed tomography (CT) scans and magnetic resonance imaging (MRI) are critical in the proper identification of sphenoid wing meningioma. Because these tumors originate near vital brain areas, early and precise detection is important. CT scans and MRIs give comprehensive pictures of the brain, allowing healthcare practitioners to determine the existence, location, and features of sphenoid wing meningiomas. Their use in the diagnostic process is critical for accurate treatment planning and improved patient outcomes. Imaging investigations are critical for identifying meningiomas, which are often dural-based tumors with isoattenuating to slightly hyperattenuating characteristics. They brighten uniformly and powerfully following iodinated contrast material injection. Meningiomas can compress the brain without entering it, and many meningiomas might be difficult to distinguish from metastases. Enhanced MRIs are critical for detecting tumor vascularization and encroachment on important vascular systems. Endovascular angiography aids in identifying tumor vascularization and patency, whereas late venous pictures are critical in determining dural sinus patency. New research technologies, such as positron emission tomography (PET), octreotide-PET, and magnetic resonance spectroscopy (MRS), have been employed to predict the aggressiveness of meningiomas in vivo.[40]

3.2 | Ophthalmic impairments

The sphenoid wing is a complex anatomical region located in the middle cranial fossa, housing critical structures such as the optic nerve, oculomotor nerve (cranial nerve III), trochlear nerve (cranial nerve IV), abducens nerve (cranial nerve VI), and the adjacent cavernous sinus. This region also includes the bony orbits that accommodate the eyeballs and their associated muscles. The location of the meningioma is pivotal in determining the specific ophthalmic impairments experienced by the patient. For instance, a meningioma at the base of the skull, near the optic nerve (CN II), can lead to visual disturbances and even blindness if left untreated. The manifestation of vision loss in the left eye and the accompanying headache in Case 2 aligns with the tumor's location in the left frontotemporal area, indicating probable involvement of the optic nerve and adjacent structures. Meningiomas affecting the anterior clinoid process, or the sphenoid ridge can encroach upon the optic canal, leading to optic nerve compression. The resultant optic neuropathy can cause progressive visual deterioration.[41] The left eye headache might be indicative of increased intracranial pressure due to the tumor's mass effect, potentially impacting the optic nerve head. On fundoscopy, the clinical findings of a pale white disc, also known as optic disc pallor, indicate a reduction in the optic nerve's blood supply and consequent damage to the nerve fibers. Tumors encroaching upon the cavernous sinus can affect multiple cranial nerves simultaneously, causing a combination of symptoms like diplopia, ptosis, and proptosis. Although explicit ophthalmic symptoms were not delineated in Case 1, the tumor's location in the left sphenoid wing raises concerns about its potential impact on the ophthalmic nerve (CN V1). Meningiomas in the sphenoid wing region can impinge upon the ophthalmic nerve (CN V1), resulting in facial numbness and potentially affecting the corneal reflex.[42] While explicit ophthalmic complaints were not stated in Case 3, the tumor's location near the foramen magnum can influence the lower cranial nerves (IX, X, XI, and XII) and the brain stem and might affect the vestibular nuclei, disrupting vestibulo-ocular reflexes and causing nystagmus, an involuntary eye movement.[43]

3.3 | Neurological impairments

The neurological symptoms observed in these cases can be attributed to two primary mechanisms: mass effect and vascular compromise. Mass effect occurs when the tumor's physical presence displaces or compresses adjacent brain tissue, leading to functional deficits. For

instance, large tumors, as in Case 2, can distort normal brain anatomy, causing headaches and seizures. Vascular compromise occurs when the tumor obstructs blood vessels, disrupting the brain's blood supply. This can lead to ischemia, causing focal neurological deficits. Additionally, tumors near critical brain areas, such as language or motor centers, can disrupt specific functions when infiltrating or compressing these regions. Focal neurological deficits, such as limb weakness (Case 1), aphasia (Case 1), and positive Hoffmann response (Case 3), are consequences of the tumor compressing or infiltrating specific brain regions. In Case 1, the left sphenoid wing meningioma led to weakness on the right side of the body due to its pressure on the motor cortex or adjacent white matter tracts. Aphasia in the same case could have resulted from the tumor encroaching upon language centers in the left hemisphere. The observation of right-sided hemiparesis and aphasia underscores the tumor's impact on motor and language centers. The meningioma's encroachment upon the frontal and temporal lobes—critical for motor control and language processing, respectively—offers a plausible explanation for the motor deficits and language impairment. The tumor's mass effect, coupled with the potential compression of adjacent white matter tracts and vascular structures, could compromise the functional integrity of these areas. The motor deficits further implicate the involvement of corticospinal pathways, as indicated by the patient's right-sided paralysis in case 2.

Hoffmann's response indicated spinal cord compression at the foramen magnum, affecting upper motor neuron pathways and resulting in hyperreflexia.[44] In Case 2, the left-sided headache likely resulted from increased intracranial pressure in the left frontotemporal area. The meningioma's size likely engendered a mass effect, precipitating increased intracranial pressure and resultant distortion of normal cerebral dynamics. Headaches could also be linked to the tumor's vicinity to meningeal and dural structures, which may compress meningeal blood vessels or induce inflammation or irritation of pain-sensitive structures in the brain's covering layers.[45] Seizures represent a prevalent symptom associated with intracranial meningiomas.[46] Seizures, as seen in Case 2, often stem from the meningioma's irritative effect on the brain cortex. In this case, the tumor might have infiltrated or pressed against the brain's motor or sensory areas, triggering abnormal electrical discharges. Peritumoral edema as seen in cases 1 and 2 has been identified as a potential factor contributing to seizures in meningiomas and could be linked to continued seizures following surgical resection.[47] Such seizures are influenced by intricate factors at the morphological, biochemical, and metabolic levels. Alterations in the peritumoral cortex involve shifts in neuronal connections, synaptic vesicle positioning, and modifications in ion channels and receptors, enhancing excitatory synapses while decreasing inhibitory ones. Biochemically, there is an elevation in Glutamatergic neurons and a reduction in GABAergic somatostatin immunoreactive neurons. Ionic imbalances encompass low Mg2+, high extracellular K+, high Fe3+, and decreased neuron-specific K+/Cl − cotransporter-2 (KCC2).[48] The tapestry of neurological symptoms underscores the multifaceted nature of such tumors and highlights the necessity of an integrative approach when elucidating the intricate interactions between tumor dynamics and resultant clinical phenotypes.

3.4 | Surgical approach

The cases highlight the importance of employing advanced techniques such as intraoperative mapping, embolization, and meticulous dissection to achieve maximal safe resection while preserving critical neural pathways. A craniotomy is deemed suitable regardless of the tumor size.[49] The objective of the surgery is to completely remove the pathological entity.[50] The surgical strategy applied to address the sphenoid wing meningioma in case 1 and case 2 involved a pterional craniotomy which provided direct access to the tumor within the sphenoid wing and allowed unobstructed visualization and manipulation of the tumor while minimizing cerebral manipulation. Meningiomas located at the foramen magnum present unique challenges due to their proximity to the medulla oblongata, lower cranial nerves, and vertebral artery, demanding careful considerations in their management. The posterior midline approach is widely recognized as the optimal choice for such meningiomas due to its lower morbidity rate compared to posterio-lateral or anterio-lateral approaches.[51] Thus, this approach was applied in case 3. Preoperative embolization can help minimize intraoperative bleeding and mitigate postoperative complications.[52] Complementing this, the strategic use of microsurgical techniques minimized the likelihood of inadvertent damage to vital neural structures, thus allevating the potential for postoperative functional deficits. The choice of the surgical approach was heavily influenced by the tumor's proximity to critical structures, including the optic nerve and surrounding neural components. Furthermore, considerations of the tumor's histopathological characteristics, such as grade and vascularity, along with projections of adhesions to neighboring structures, significantly shaped the approach.

3.5 | Intraoperative findings

In Case 1, the presence of the unexpected, thick-walled abscess required careful excision and subsequent management to prevent infection. Despite this, a near-total resection was achieved, but due to the tumor's invasive nature, complete removal was not possible. Several factors described in[53] may make meningiomas prone to abscess formation. These include their high vascularity and unique blood flow pattern. The absence of the blood–brain barrier and a lack of immune response further contribute, creating an environment conducive to abscess development within these tumors. In Case 2, the decision to perform preoperative embolization was crucial in minimizing bleeding during surgery. Embolization induces vascular loss in the target area, promoting necrosis in the targeted lesion before surgery and easing the resection process.[54] In Case 3, the bone-invasive growth pattern highlighted the potential for incomplete resection, influencing the long-term recurrence rates and overall outcomes, emphasizing the importance of a multidisciplinary approach for managing symptoms postsurgery.[55] Overall, these findings underscore the necessity of adaptability and strategic decision-making in such neurosurgical procedures.

3.6 | Long-term follow-up

This study[56] found that a shorter time from diagnosis to surgery, larger tumor size, and compression were predictors of postoperative improvement. Among these factors, compression was identified as an independent predictor, indicating its significant influence on the postoperative improvement in motor control and strength. In the postoperative assessment of this study,[57] patients commonly experienced challenges such as double vision, hearing loss, balance problems, and reduced sensation in the V1 and V2 cranial nerve areas. However, it was notable that most patients had developed adaptive strategies to cope with these difficulties. After surgery in our cases, the patients received thorough postoperative care and were closely monitored for complications like infections or neurological deficits. Long-term follow-ups included regular assessments for late complications and periodic MRI scans to detect any tumor recurrence or progression as meningiomas of the sphenoid ridge constitute about 25% of recurring meningiomas.[58]

4 | CONCLUSION

In conclusion, the presented cases shed light on the intricate challenges posed by meningiomas, particularly in their significant impact on both ophthalmic and neurological functions. The diverse spectrum of symptoms, ranging from visual disturbances to focal neurological deficits, underscores the complexity of these tumors and the diversified ways they affect patients. These cases emphasize the crucial role of a multidisciplinary approach in managing meningiomas effectively. The joint effort of neurosurgeons, ophthalmologists, and other specialists is paramount. Such that their combined expertise ensures a holistic understanding of each case, leading to tailored treatment approaches. This collaboration not only enhances the precision of surgical interventions but also facilitates comprehensive postoperative care and long-term follow-up.

AUTHOR CONTRIBUTIONS

Abdullah Nadeem: Conceptualization; data curation; formal analysis; project administration; writing – original draft; writing – review and editing. **Afsheen Khan:** Conceptualization; writing – original draft; writing – review and editing. **Ashna Habib:** Conceptualization; writing – original draft; writing – review and editing. **Rabeea Tariq:** Writing – original draft; writing – review and editing. **Areeba Ahsan:** Writing – original draft; writing – review and editing. **Areeba Aamir Ali Basaria:** Writing – original draft; writing – review and editing. **Nahid Raufi:** Funding acquisition; writing – review and editing. **Abir Chughtai:** Writing – original draft; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

There are no conflicts of interest.

DATA AVAILABILITY STATEMENT

Not applicable.

CONSENT

Written informed consent was obtained from each patient to publish this series in accordance with the journal's patient consent policy.

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